

AMENDMENT AND RESPONSE TO OFFICE ACTION UNDER 37 C.F.R. § 1.116

In the Claims

1. (Previously presented) A pharmaceutical composition comprising a compound selected from the group shown in Table 1, which specifically alters the binding activity of SR-BI, in combination with a pharmaceutically acceptable carrier.

2. (Previously presented) The composition of claim 1 in a dosage formulation comprising an amount effective to treat a human or animal in need thereof.

3. (Previously presented) The composition of claim 1, wherein the compound is selected from the group consisting of BLT-1 (MIT 9952-53), BLT-2 (MIT 9952-61), BLT-3 (MIT 9952-19), BLT-4 (MIT 9952-29), and BLT-5 (MIT 9952-6).

4. (Previously presented) A method for altering cholesterol transport into or out of cells comprising inhibiting expression or activity of SR-BI comprising administering to an animal or human in need thereof a pharmaceutical composition comprising a compound selected from the group shown in Table 1, which specifically alters the binding activity of SR-BI, in combination with a pharmaceutically acceptable carrier.

5. (Currently amended) The method of claim 4, wherein the ~~composition of claim 1 pharmaceutical composition~~ enhances HDL binding by increasing SR-BI's binding affinity for HDL.

6. (original) The method of claim 4, wherein the inhibited SR-BI binding activity blocks SR-BI-mediated lipid transport.

7. (original) The method of claim 6, wherein the inhibited SR-BI binding activity blocks SR-BI-mediated selective lipid uptake.

AMENDMENT AND RESPONSE TO OFFICE ACTION UNDER 37 C.F.R. § 1.116

8. (original) The method of claim 7, wherein the lipid is HDL cholesteryl ether.

9. (original) The method of claim 4, wherein the inhibited SR-BI binding activity blocks efflux of cellular cholesterol to HDL.

10. (currently amended) A method of identifying a compound which alters SR-BI binding activity or expression comprising screening a library of small molecule compounds using a high throughput screening assay determining alteration of HDL binding by SR-BI[,] or SR-BI-mediated lipid transport ~~or expression of SR-BI~~.

11. (Previously presented) The method of claim 10, wherein the SR-BI expression is determined by Northern blot analysis.

12. (original) The method of claim 10, wherein the library is a chemical library.

13. (original) The method of claim 10, wherein the SR-BI binding activity is inhibited.

14. (original) The method of claim 13, wherein the inhibited SR-BI binding activity blocks SR-BI-mediated lipid transport.

15. (original) The method of claim 14, wherein the inhibited SR-BI binding activity blocks SR-BI-mediated selective lipid uptake.

16. (original) The method of claim 15, wherein the lipid is HDL cholesteryl ether.

17. (original) The method of claim 10, wherein the inhibited SR-BI binding activity blocks efflux of cellular cholesterol to HDL.